matory changes (Fig. 3). Electromyographic study was not done and the C.S.F. was not examined.

The patient was submitted to irradiation of the mediastinum and left axilla. Treatment was supplemented by prednisolone (15–30 mg. daily). The mediastinal masses regressed completely and the patient's general condition improved. The muscular weakness, however, remained unchanged. He was discharged home on 15 October, but on 11 February 1963 he had to be readmitted owing to deterioration of his muscular weakness. On examination the weakness of the limbs was now more severe; there was, in addition, marked weakness of the trunk and jaw muscles, bilateral ptosis, and difficulty in swallowing; his speech was very weak and his voice almost inaudible. All tendon reflexes were now absent. No clinical or radiological evidence of his primary disease was present. He was discharged on 18 February and died at home a few weeks later. Necropsy was not performed.

## COMMENTS

Åström *et al.* (1958) and Cavanagh *et al.* (1959) reported demyelinating conditions of the brain arising in the course of Hodgkin's disease or chronic lymphatic leukaemia. Lloyd and Urich (1959) described a case of acute disseminated demyelination of the brain associated with lymphosarcoma.

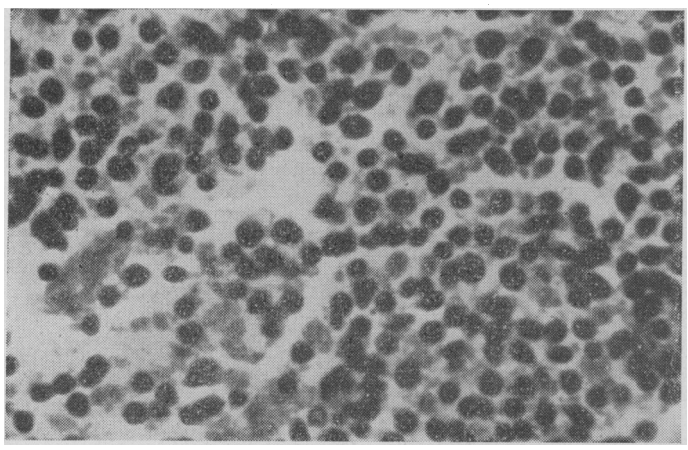

FIG. 2.—Section of lymph node (×250). The normal architecture of the node has been destroyed by the sarcomatous cells.

Lymphosarcoma and reticulum-cell sarcoma complicated by polyneuropathy has been reported by Holt (1961), while Rooke *et al.* (1960) found one case of reticulum-cell sarcoma among

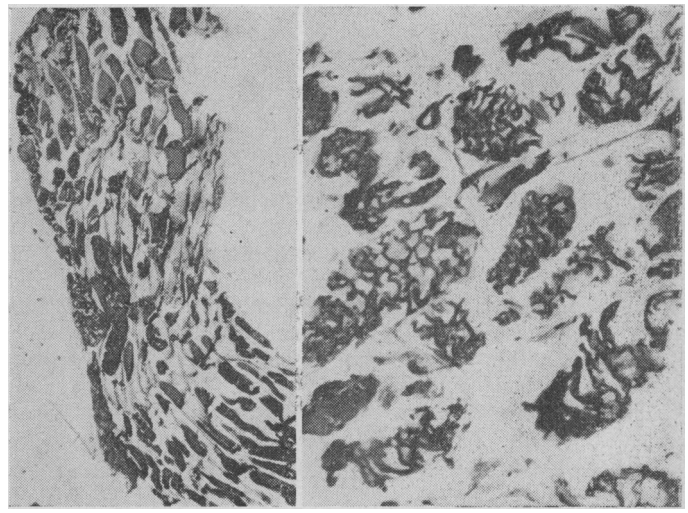

FIG. 3.—Section of the quadriceps muscle showing marked fragmentation and vacuolation of the muscle fibres. (Left, ×15; Right, ×60.)

19 patients with carcinomatous myasthenic syndrome associated with malignancy.

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# Oxygen Therapy in Cyanide Poisoning

*Brit. med. J.*, 1964, **2**, 33–34

Survival after accidental ingestion of cyanide and its salts is not unknown (Polson and Tattersall, 1959; Graham, 1962), but in most cases, though not all, the amount of cyanide has been small and probably less than the lethal dose. This case is recorded because the amount of cyanide taken is known and exceeds the accepted lethal dose of 0.5 g. (Graham, 1962) and because it illustrates the value of modern techniques of resuscitation.

Experimental work has shown that a high tension of oxygen in the tissue protects against the cell asphyxia induced by cyanide salts (Cope, 1961). Our experience suggests that even after the tissues have been invaded by cyanide hyperventilation with oxygen may be a useful therapeutic procedure.

## CASE REPORT

A 39-year-old amateur photographer had prepared 3 fl. oz. (85 ml.) of a 1% solution of potassium cyanide to "reduce" negatives. In error, he drank 2¾ oz. (78.3 ml.) of this (0.8 g. cyanide) instead of lemonade. As a result of a "999" call, he was brought into hospital less than five minutes after taking the cyanide. Deceptively pink in colour, he was deeply comatose. The systolic blood-pressure was 60 mm. Hg and the radial pulse imperceptible. Respiration was both irregular and hypopnoeic. Despite crushing amyl nitrite capsules to allow inhalation of the vapour, during transit from the casualty department to an intensive care unit in the theatre block he had two convulsions, and both respiration and detectable cardiac action ceased. External cardiac massage was performed and continued for 15 minutes until an E.C.G. showed spontaneous contractions occurring. Meanwhile a tracheostomy was performed and a cuffed tube sealed into the trachea. Using a Boyle's apparatus, artificial respiration was carried out with pure oxygen at a rate of 16 "breaths" per minute. Hydrocortisone ("efcortelan") 100 mg. was given by intracardiac injection at the time of apparent cardiac arrest, and repeated twice during the recovery period when extreme slowing of the heart recurred (Fig. 1). Artificial respiration was discontinued after 90 minutes when adequate spontaneous respiratory efforts were obvious.

After this, sodium thiosulphate (25 g. in 50 ml.) and sodium nitrite (0.3 g. in 10 ml.) were obtained, and, though recovery seemed likely as a result of the measures already taken, these drugs were given by intravenous injection to assist the clearing of cyanide from the tissues. The patient recovered consciousness four hours after admission. Apart from amnesia for 24 hours after his collapse he showed little upset from his experience and was discharged from hospital after 11 days.

## COMMENT

Cyanide owes its extreme toxicity to chelation of the metallic moiety of cytochrome oxidase and other intracellular enzymes concerned in the utilization of oxygen in the tissues. This results in cellular asphyxia, and because of this feature of the poison the blood remains oxygenated until respiration fails. The bulbar centres are rapidly paralysed after a period of excitation manifest by hypopnoea (Cope, 1961) and alternating tachycardia and bradycardia (Wexler, Whittenberger, and Dumke, 1947). Vomiting and convulsions are terminal events (*Lancet*, 1961).

The work of Chen and Rose (1952) established sodium nitrite and thiosulphate as effective agents in treatment, the former to bind as much cyanide as possible as cyanmethaemoglobin and thus minimize the effect on tissues, and the latter to promote natural detoxication by the enzyme rhodonase. Arising from the experimental work of Cope (1961), which showed that hyperventilation with oxygen before a dose of cyanide had a protective effect, the suggestion was made that oxygen therapy might be of value in treatment (*Lancet*, 1961).

In this case oxygen therapy was started after the "terminal events" of convulsions and apparent failure of vital functions. Despite this, it was possible to reverse the terminal stages as seen in the E.C.G. (Fig. 2) and described by Wexler *et al.* (1947)

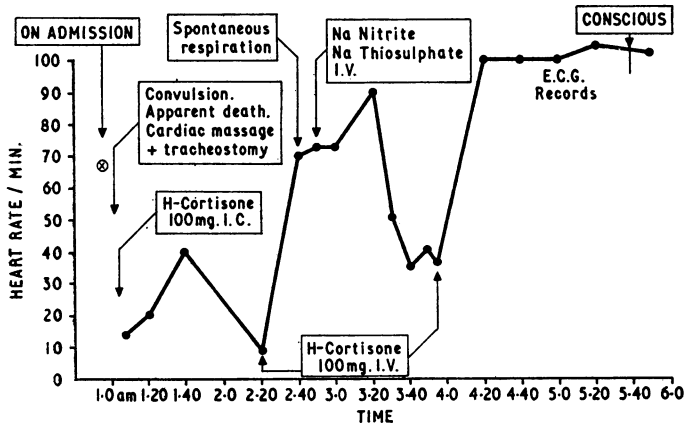


FIG. 1.—Effect of drugs given during treatment.

and obtain recovery. Though it would appear from the timing that the improvement was related more closely to the hydrocortisone injections (Fig. 1), these were given primarily as supportive therapy and it seems more reasonable to suppose that resuscitation was chiefly due to the continued administration of oxygen and artificial hyperventilation at the critical early stages.

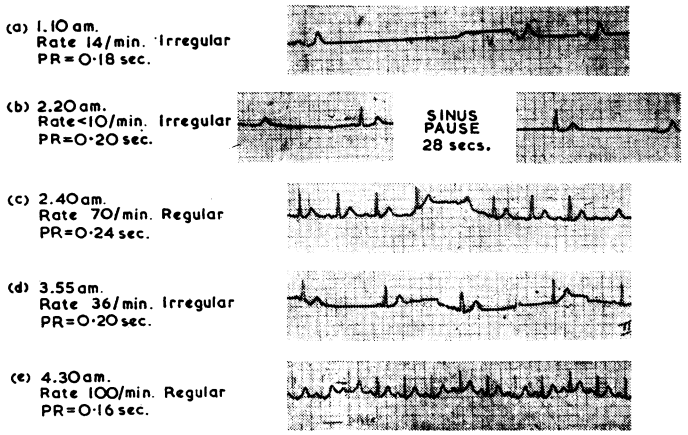


FIG. 2.—E.C.G. tracings taken during treatment.

While no conclusions can be drawn from a single experience of this kind of emergency, we have been left with the impression that, though potassium cyanide is a rapidly active tissue poison, the effect on cellular metabolism may be more transient than has been appreciated, and if vital functions are maintained even in the face of apparent death recovery may be possible.

We are grateful to Dr. A. A. F. Peel, in whose wards this patient was treated, for permission to publish this case report.

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